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ORIGINAL RESEARCH

Diagnostic and prognostic value of optic nerve sheath diameter in methanol intoxication

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Abstract

Background: Methanol intoxication can lead to death, and early differential diagnosis and treatment are of paramount importance. In this study, patients with ethanol and methanol intoxication were compared in terms of optic nerve sheath diameter (ONSD). We also examined ONSD as a diagnostic parameter and a predictor of mortality. **Methods**: This retrospective study included patients who presented to the emergency department due to alcohol consumption and divided them according to the presence of methanol intoxication versus no methanol intoxication. Demographic data, vital parameters, ONSD values, need for dialysis, visual impairment, and survival were recorded. **Results**: Mean ONSD in the methanol group was significantly higher than in the ethanol group ($p < 0.001$). In the receiver-operating characteristic (ROC) curve analysis conducted to evaluate the predictive power of ONSD in distinguishing methanol intoxication from ethanol intoxication, an ONSD cut-off value of 5.57 mm was found to have a sensitivity of 85.2% and a specificity of 80.0% (Area under the curve (AUC) = 0.887; 95% confidence interval (CI) = 0.801–0.973; $p < 0.001$). **Conclusions**: The results obtained in the current study showed that ONSD value is high in methanol poisoning cases and can be used in the differential diagnosis of patients presenting with alcohol consumption and suspected methanol toxicity.

Keywords

Alcohol; Ethanol; Methanol; Optical disk; ONSD

1. Introduction

Methanol, commonly referred to as wood alcohol or methyl alcohol, is a transparent, colorless, and flammable liquid characterized by a sweet, syrupy smell. It is primarily obtained via the distillation of wood or synthesized chemically from natural gas or coal [1]. It is particularly common in developing countries and in countries where alcohol consumption is prohibited. Methanol causes toxicity and its intoxication usually happens as a result of consuming contaminated alcohol produced from illegal domestic production [2, 3]. Methanol consumption is associated with high morbidity and mortality [4]. Mortality rates have been reported to range between 18% and 44% [5]. The metabolism of methanol in humans is primarily facilitated by the enzymes alcohol dehydrogenase and aldehyde dehydrogenase (ALDH). Alcohol dehydrogenase initiates the process by converting methanol into formaldehyde, which is subsequently oxidized to formic acid—a highly toxic compound—through the action of ALDH. In humans, the metabolic clearance of formic acid is inefficient, resulting in its accumulation and associated toxicity. Formic acid disrupts cellular metabolism by depleting adenosine triphosphate (ATP) levels and inhibiting oxidative phosphorylation, primarily through its interaction with cytochrome c oxidase, a critical enzyme in the mitochondrial respiratory chain [6, 7]. This impairment of oxidative phosphorylation promotes lactate accumulation, while the buildup of formic acid contributes to high anion gap metabolic acidosis. Additionally, elevated molar concentrations of methanol further aggravate the condition by increasing the osmolal gap. The systemic effects of methanol intoxication typically include central nervous system (CNS) depression, visual disturbances and metabolic acidosis. Initial symptoms—such as abdominal discomfort, vomiting, nausea, and headache—often arise within the first four hours post-ingestion, whereas more severe manifestations, including CNS impairment and visual loss, tend to occur approximately 12 hours after exposure [3, 6, 8].

Methanol exposure leads to damage of the optic nerve and retina. Although the minimum volume required to cause permanent visual impairment in adults remains uncertain, it is generally considered to exceed 30 mL. Following oral ingestion, methanol can induce permanent and complete vision loss within 10 to 30 hours, a process that may be prolonged if ethanol is co-ingested [9]. Visual disturbances such as blurred

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vision, diplopia, photophobia, and altered color perception are reported in approximately 25–33% of methanol poisoning cases. Moreover, patients may experience visual field constriction and, in some instances, total vision loss. Ophthalmologic examination may reveal pupillary dilation, loss of pupillary reflexes, optic disc hyperemia, pallor, or edema [10, 11]. These manifestations result from retrobulbar axonal demyelination, a progressive pathology culminating in optic nerve atrophy. The optic nerve sheath, a membrane enveloping the optic nerve posteriorly and continuous with the cranial dura mater, can show optic disc edema in the acute phase, detectable via imaging modalities such as ocular computed tomography (OCT) and magnetic resonance imaging (MRI) [3, 12]. However, no studies to date have investigated optic nerve sheath diameter measurement by brain computed tomography for differential diagnosis in patients presenting with alcohol consumption.

This study represents the first investigation into alterations of optic nerve sheath diameter (ONSD) on brain computed tomography in patients presenting to the emergency department following ethanol or methanol ingestion.

2. Methods

2.1 Study setting and study population

This retrospective study included patients who presented to the emergency department of a tertiary care hospital for alcohol consumption between 01 October 2018 and 01 October 2023. The study group comprised individuals diagnosed with methanol intoxication, whereas the control group included patients with confirmed positive ethanol levels and absence of acidosis on blood gas analysis.

Data were collected by reviewing hospital electronic records and patient files. International Classification of Diseases (ICD) 10 diagnosis codes “T51.1, T51.8 and T51.9” were used to search for relevant patients. When determining whether patients were suitable for the study, symptoms, clinical evaluation and supportive laboratory tests were used since the methanol test was not checked.

Methanol group (MG) included patients diagnosed with methanol poisoning based on clinical and laboratory evaluation, as methanol levels could not be measured in our hospital. As reported by Roberts DM *et al.* [13], in situations where direct methanol measurement is unavailable, the presence of high anion gap metabolic acidosis or elevated osmolal gap, combined with clinical suspicion—such as coma, seizures, or new-onset visual impairment—can assist in diagnosis. In our study, a pH threshold of <7.30 was used to define metabolic acidosis. The ethanol group (EG) consisted of patients with positive ethanol levels (measured range: 10.1–498 mg/dL; positivity defined as >10.1 mg/dL) and absence of acidosis on blood gas analysis.

The study population comprised patients meeting the inclusion criteria who presented to the emergency department due to alcohol consumption, either as walk-in outpatients or transported by ambulance (112 service).

Due to the high prices of alcohol in our country, methanol is used both in other ethanol-containing substances (cologne, hand sanitizer, *etc*.) and in sectors such as illegal alcohol production. Our clinic is a center that frequently receives applications with such confusion after drinking alcohol. However, in addition to this; since these patients have confusion upon arrival and usually do not have companions, a clear anamnesis cannot be obtained. Since we can not exclude the finding of head trauma in these patients, as included in the Nexus criteria [14], cranial computed tomography (CT) is performed for the differential diagnosis of confusion (especially in patients with a Glasgow coma scale <14) and because of abnormal behaviors in these patients. As stated in our exclusion criteria, the fact that we see intracranial hemorrhage and mass in some of these patients clarifies our situation. Therefore, cranial imaging is recommended in patients admitted with similar symptoms in our clinic.

Pregnant patients, individuals with incomplete data, those with head trauma, intracranial hemorrhage, or space-occupying lesions, patients with non-measurable optic nerve sheath diameter (ONSD), absence of brain CT imaging, as well as those with pre-existing visual impairments or ocular diseases affecting ONSD were excluded from the study. A total of 42 adult patients with methanol intoxication and 30 adults with ethanol intoxication who met the inclusion criteria and were aged 18 years or older were included. Five patients were excluded due to intracranial hemorrhage, five due to head trauma, three for incomplete data, and two due to intracranial masses.

Patient enrollment for this study was conducted consecutively, without employing randomization or blinding methods. Instead, all patients presenting to our emergency department within the defined time frame and met the inclusion criteria were consecutively included in the study.

Comprehensive patient data were collected retrospectively from the Hospital Information Management System and recorded using a standardized case report form. Variables included demographic information (age, gender), vital signs, right and left optic nerve sheath diameter (ONSD) measurements obtained from brain CT scans, clinical outcomes such as admission or discharge status, in-hospital mortality, and the need for dialysis. Additionally, the presence of visual impairment, blood gas analysis parameters, time elapsed since alcohol consumption, as well as serum urea and creatinine levels were documented. Data were recorded in the case record form by 3 emergency medicine specialists working in the emergency department via the patient information management system. Emergency medicine specialists had at least 3 years of experience. In case of any discrepancy in the collected data, it was reviewed again by a 3 specialist and the final decision was made by consensus. The results of the first blood samples taken at the emergency room presentation were used. Blood samples from patients were promptly sent to the laboratory following collection, and all specimens were centrifuged at $3000 \times g$ for 10 minutes at 25 ºC. Then, the samples were analyzed without waiting using the Roche brand Ethanol Gen.2 kit (plasma/serum) (Roche Diagnostics, Rotkreuz, ZG, Switzerland), calibrator and controls using the alcohol dehydrogenase enzymatic method. The measurement range of the kit is 10.1–498 mg/dL.

2.2 Measurement of the ONSD

Optic nerve sheath diameter (ONSD) measurements were obtained from the initial brain CT scans conducted upon emergency department admission. Non-contrast head CT scans were performed using a standardized brain CT protocol with 1-mm contiguous slices. All measurements utilized uniform window width (WW 40), window level (WL 250), contrast, and brightness settings. The ONSD was measured 3 mm posterior to the globe, just beneath the sclera. To ensure accuracy, measurements were taken on the coronal plane, assessing the transverse diameter of the ONSD. All measurements were conducted by a radiologist under double-blind conditions.

2.3 Statistical analyses

Statistical analyses were conducted using SPSS version 26.0 for Windows® (IBM Corp., Chicago, IL, USA). Descriptive statistics were expressed as frequencies, percentages, means with standard deviations, medians, and ranges (minimum–maximum). The Kolmogorov-Smirnov test assessed data normality. Categorical variables were compared using the Pearson chi-square test, while Fisher's Exact test was applied when group sizes were below five. For continuous variables, Student's *t*-test was employed for normally distributed data, and the Mann-Whitney U test was used for non-parametric comparisons. Receiver operating characteristic (ROC) curve analysis evaluated the diagnostic performance of ONSD in differentiating methanol from ethanol intoxication, with area under the curve (AUC), optimal cut-off values, sensitivity and specificity calculated. Correlation analyses assessed relationships between ONSD, patient age, and time elapsed between ingestion and hospital admission. Furthermore, binary logistic regression was performed to estimate odds ratios for mortality risk, dialysis requirement, and visual impairment associated with variations in ONSD. Statistical significance was defined as $p < 0.05$ with a 95% confidence interval.

3. Results

The study included a total of 57 patients, consisting of 27 patients with methanol intoxication (methanol group—MG) and 30 patients with ethanol intoxication (ethanol group—EG). There was only one female patient in the EG. Age, gender and vital parameters of the cases are given in Table 1.

When evaluating the time from alcohol ingestion to presentation at the emergency department, the mean time was 12.00 $\pm$ 9.8 hours in the MG and 5.73 $\pm$ 3.02 hours in the EG, and this duration was statistically significantly longer in the MG ($p = 0.002$). In both the MG and EG, brain CT imaging was used to measure the right and left ONSDs, and the arithmetic mean of the obtained values was used to determine the ONSD. The results showed that mean ONSD was significantly higher in the MG compared to the EG ($p < 0.001$) (Table 1).

Significant differences were observed between the methanol group (MG) and the ethanol group in several laboratory parameters. Specifically, pH, bicarbonate (HCO_3), and partial pressure of carbon dioxide (PCO_2) levels were markedly lower in the MG ($p < 0.001$, $p < 0.001$, and $p = 0.001$, respectively), whereas lactate, creatinine, and base deficit (BD) levels were significantly elevated ($p = 0.007$, $p < 0.001$, and $p < 0.001$, respectively). No significant difference was detected in urea levels between the groups (Table 1). The requirement for hemodialysis was significantly greater in the MG ($p < 0.001$), accompanied by a higher incidence of visual impairment ($p < 0.001$). Mortality was exclusive to the methanol-intoxicated patients within the MG, with this difference reaching statistical significance ($p = 0.001$) (Table 1).

ONSD values were further analyzed according to hemodialysis necessity, presence of visual impairment, and mortality status. Results indicated that mean ONSD was significantly elevated in patients requiring hemodialysis, those exhibiting visual impairment, and those who died (Table 2). Receiver operating characteristic (ROC) curve analysis assessing the diagnostic accuracy of ONSD in differentiating methanol from ethanol intoxication revealed an optimal cut-off value of 5.57 mm, yielding a sensitivity of 85.2% and specificity of 80.0% (AUC = 0.887; 95% CI: 0.801–0.973; $p < 0.001$) (Fig. 1).

Correlation analysis demonstrated a moderate positive relationship between mean ONSD and the elapsed time from alcohol ingestion to hospital presentation ($r = 0.440$; $p = 0.001$). Furthermore, logistic regression analysis revealed that each 1 mm increase in ONSD was associated with a 1.94-fold increase in mortality risk, a 2.56-fold increase in risk of visual impairment, and a 2.44-fold increase in the necessity for dialysis.

4. Discussion

This study demonstrated that optic nerve sheath diameter measurement may serve as a useful tool for differentiating methanol intoxication in patients presenting to the emergency department with a history of alcohol consumption. Additionally, higher ONSD levels were observed in patients with visual impairment at presentation, and higher ONSD measurements were associated with higher mortality in patients with alcohol intoxication. Although there are several studies in the literature in which ONSD was measured by ultrasonography, we did not find any studies that used brain computed tomography for ONSD measurement. Therefore, we believe that the present study will contribute to the literature.

Kaewput *et al.* [15] reported that 70% of methanol-intoxicated patients were male, with a mean age of 38 $\pm$ 18 years. Similarly, all patients with methanol intoxication in the current study were male, but with a higher mean age of 54.26 $\pm$ 11.55 years. The demographic data of our study indicate both a higher mean age and a predominance of male patients. The observed age difference in our study could be due to demographic and cultural differences.

Methanol ingestion is associated with high mortality and morbidity. In a study by Aghababaeian *et al.* [16] on the 2018 methanol outbreak in Iran *involving* 768 patients, the mortality rate was reported as 10.1%. In a study by Rahimi *et al.* [5], it was reported that methanol intoxication has a mortality rate ranging from 18% to 44%. In their study, Güler *et al.* [17] reported a mortality rate of 46.5%. Studies in the literature have clearly reported a wide range of mortality rates. We believe that these variations in mortality rates are mainly

TABLE 1. Examination of demographic, laboratory and clinical data of patients in MG and EG.

Parameter	All cases (n = 57) n (%)/Mean ± SD	Methanol (n = 27) n (%)/Mean ± SD	Ethanol (n = 30) n (%)/Mean ± SD	*p**
Age (yr)	49.65 ± 13.87	54.26 ± 11.55	45.50 ± 14.63	0.016
Gender				
Male	56 (98.2)	27 (100.0)	29 (96.7)	0.942
Female	1 (1.8)	0 (0.0)	1 (3.3)	
Blood pressure at presentation				
Systolic BP	121.25 ± 22.68	120.18 ± 28.52	123.47 ± 13.85	0.914
Diastolic BP	74.53 ± 15.89	76.00 ± 20.97	72.40 ± 8.50	0.318
Time between alcohol ingestion and presentation	8.70 ± 7.70	12.00 ± 9.8	5.73 ± 3.02	0.002
Optic nerve sheath diameter	5.65 ± 0.73	6.14 ± 0.64	5.20 ± 0.47	<0.001
Hemodialysis performed				
Yes	29 (50.9)	27 (100.0)	2 (6.7)	<0.001
No	28 (49.1)	0 (0.0)	28 (93.3)	
Laboratory values				
pH	7.21 ± 0.25	7.01 ± 0.23	7.39 ± 0.05	<0.001
HCO_3	17.37 ± 10.09	8.19 ± 6.27	25.62 ± 3.49	<0.001
Lactate (mg/dL)	3.73 ± 3.91	5.17 ± 5.22	2.44 ± 1.18	0.007
PCO_2	37.93 ± 13.13	31.87 ± 15.89	43.37 ± 6.47	0.001
Base deficit	−10.24 ± 13.20	−22.13 ± 9.22	0.46 ± 3.16	<0.001
Urea (mg/dL)	26.05 ± 10.36	27.89 ± 12.88	24.40 ± 7.24	0.207
Creatinine (mg/dL)	0.95 ± 0.37	1.22 ± 0.37	0.72 ± 0.15	<0.001
Visual impairment				
Yes	23 (40.4)	23 (85.2)	0 (0.0)	<0.001
No	34 (59.6)	4 (14.8)	30 (100.0)	
Mortality				
Yes	9 (15.8)	9 (33.3)	0 (0.0)	0.001
No	48 (84.2)	18 (66.7)	30 (100.0)	

**: Pearson χ^2 Test, Fisher's Exact Test, and Independent Samples T Test were used. HCO_3: bicarbonate; PCO_2: partial pressure of carbon dioxide; SD: Standard deviation; BP: Blood pressure.*

TABLE 2. Comparison of ONSD measurements according to key demographic and clinical data of the patients.

Parameter	Optic nerve sheath diameter (mm) Mean ± SD	*p*
Hemodialysis performed		
Yes	6.06 ± 0.68	<0.001
No	5.21 ± 0.49	
Visual impairment		
Yes	6.19 ± 0.64	<0.001
No	5.28 ± 0.53	
Mortality		
Yes	6.43 ± 0.48	<0.001
No	5.50 ± 0.67	

SD: Standard deviation.

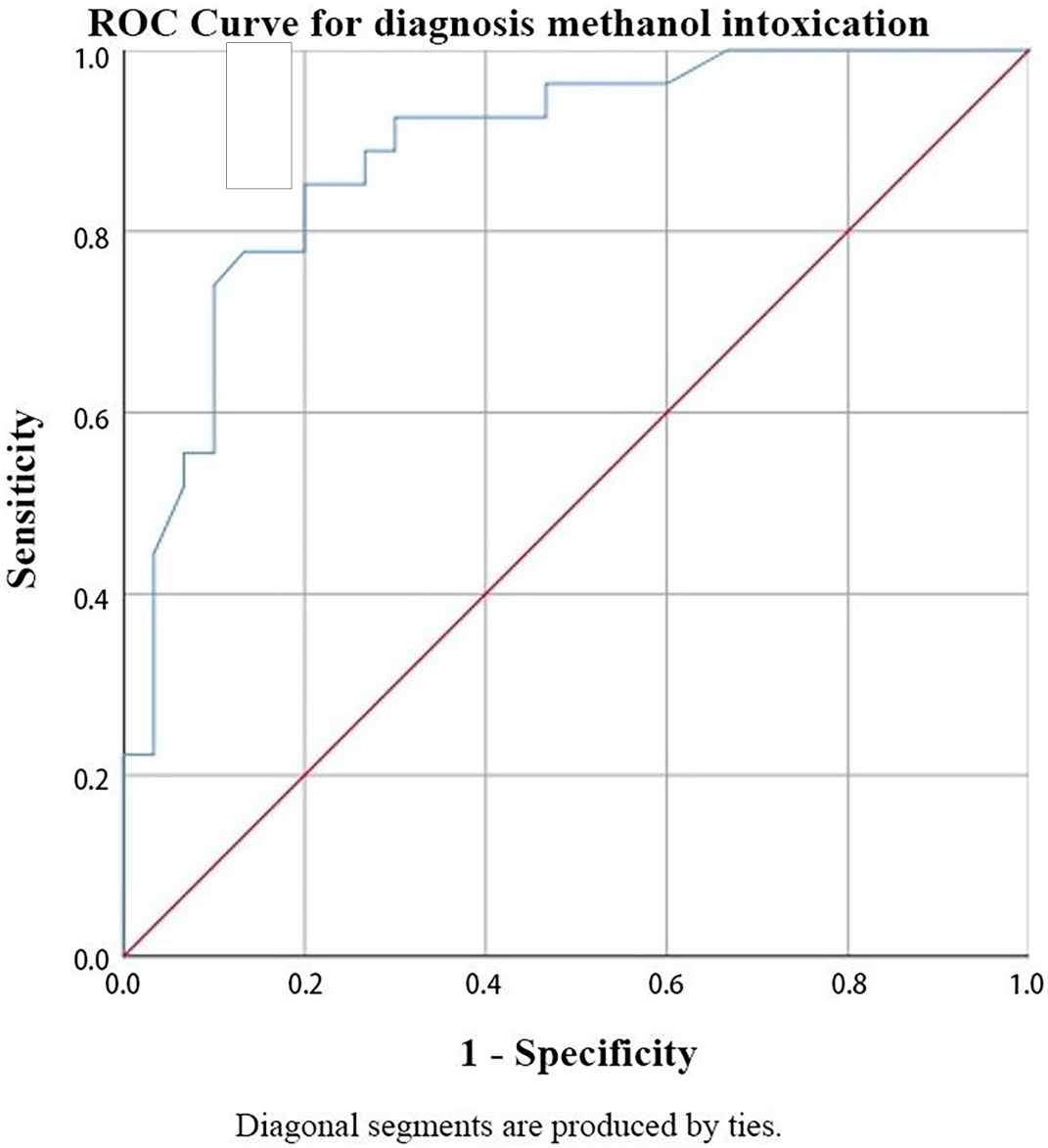

FIGURE 1. Evaluation of the success of ONSD values in differentiating methanol and ethanol intoxication. ROC: Receiver operating characteristic.

due to the differences in the amount of alcohol consumed by patients, the time of presentation to the hospital, and the challenges encountered in accessing effective treatment. The results of the present study show some alignment with findings from certain studies in the literature, while presenting lower mortality rates. The lower mortality rates observed in this study may also be attributed to the exclusion of cases with intracranial hemorrhage. In this study, it was found that each 1 mm increase in ONSD is associated with a 1.94-fold increase in the risk of mortality. This implies that ONSD at the time of presentation appears to be a predictor of mortality.

The primary cause of acidosis observed in methanol intoxication is formic acid, which is a metabolite of methanol [18]. Additionally, Hovda *et al.* [19] identified increased lactic acid as another cause of acidosis, resulting from disruptions in ATP production, leading to high anion gap metabolic acidosis as a consequence. In our present study, significantly higher anion gap and lactate levels were expected between the ethanol and methanol groups. In addition, lower pH, PCO_2 and HCO_3 levels were observed in patients with methanol poisoning compared to the ethanol group. Sasani *et al.* [20] reported that the depth of metabolic acidosis was strongly associated with increased mortality.

Methanol intoxication inhibits cytochrome oxidase, resulting in impaired ATP synthesis and consequent energy deficiency, which particularly affects optic nerve cells due to their high metabolic demands. Additionally, free formic acid contributes to axonal and myelin sheath damage, leading to optic neuropathy and myelin sheath edema detectable through ONSD measurement [6, 21]. ONSD measurement is widely utilized for monitoring increased intracranial pressure in patients with head trauma or brain injury [14, 15]. Moretti *et al.*'s [22] systematic review indicated that ONSD is effective in detecting elevated intracranial pressure, although no consensus exists on the definitive threshold value. While a threshold of 5 mm is commonly accepted, Geeraerts *et al.* [23] suggested it may be as high as 5.9 mm. In the current study, an ONSD cut-off of 5.57 mm yielded a sensitivity of

85.2% and specificity of 80.0%. Furthermore, Grzybowski *et al.* [24] reported that methanol exposure damages the optic nerve and retina, causing visual impairment. Sharma *et al.* [10], in a case series of eight methanol intoxication patients, documented optic disc edema via fundoscopy. Consistent with these findings, our study found higher ONSD values in patients with visual impairment. Sener *et al.* [6] reported a cut-off of 5.05 mm for ONSD measured by ultrasonography, with sensitivity and specificity of 80.8% and 100%, respectively. Differences between these findings may be attributed to the varying measurement modalities—ultrasound versus computed tomography—and differences in cut-off values.

5. Limitations of study

This study has several limitations. Firstly, methanol concentrations could not be measured at our facility, preventing correlation analysis between methanol levels and optic nerve sheath diameter (ONSD). Additionally, the study's single-center, retrospective design and relatively small sample size represent further limitations. Nevertheless, we believe these factors do not significantly impact the study's overall findings.

However, to generalize the results of the study, it is necessary to support them with multicenter, randomized controlled studies *involving* a larger number of cases.

6. Conclusions

The findings of the present study revealed an increase in ONSD in patients with methanol intoxication, whereas no significant change was detected in those with ethanol intoxication. We propose that ONSD measurement may serve as a valuable tool both for differential diagnosis and prognostic assessment in patients presenting with alcohol consumption and suspected methanol toxicity. As the first investigation addressing this subject, the results are expected to contribute meaningfully to the existing body of literature.

AVAILABILITY OF DATA AND MATERIALS

The data that support the findings of this study are available from the corresponding author, JW, upon reasonable request.

AUTHOR CONTRIBUTIONS

TÇ, KŞ, AÇ—analyzed the data; wrote the manuscript, designed the research study; performed the research; provided help and advice on this study. GA, AT, AÖ—designed the research study; performed the research. İB, MAE—designed the research study; provided help and advice on this study. RB—designed the research study; performed the research; provided help and advice on this study. ZK—designed the research study. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article. Owing to the retrospective design of the study and the decision by the Ethics Committee of Mersin City Hospital, obtaining informed consent from patients was waived. Ethical approval was obtained from the Mersin University Ethics Committee prior to the commencement of the study (Approval No: 2024/694; Date: 24 July 2024).

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES

[1] Allata Y, Chouhani BA, El Bardai G, Kabbali N, Sqalli Houssaini T. The role of nephrologists in the management of methanol poisoning. Cureus. 2023; 15: e37471.

[2] Khalili MR, Sadati MS, Jahanbani-Ardakani H. Outbreak of methanol-induced optic neuropathy amid COVID-19 pandemic. Graefe's Archive for Clinical and Experimental Ophthalmology. 2021; 259: 1375–1376.

[3] Liberski S, Kaluzny BJ, Kocięcki J. Methanol-induced optic neuropathy: a still-present problem. Archives of Toxicology. 2022; 96: 431–451.

[4] Ashurst JV, Schaffer DH, Nappe TM. Methanol Toxicity. StatPearls: Treasure Island (FL). 2025.

[5] Rahimi M, Hosseini SM, Mohtarami SA, Mostafazadeh B, Evini PET, Fathy M, *et al*. Prediction of acute methanol poisoning prognosis using machine learning techniques. Toxicology. 2024; 504: 153770.

[6] Sener K, Cakır A, Altug E, Korkut S, Güven R, Kapci M. Is optic nerve sheath diameter diagnostic in methanol intoxication? Alcohol. 2023; 113: 27–31.

[7] Ahiskali I, Pinar CL, Kiki M, Cankaya M, Kunak CS, Altuner D. Effect of taxifolin on methanol-induced oxidative and inflammatory optic nerve damage in rats. Cutaneous and Ocular Toxicology. 2019; 38: 384–389.

[8] Alhusain F, Alshalhoub M, Homaid MB, Esba LCA, Alghafees M, Al Deeb M. Clinical presentation and management of methanol poisoning outbreaks in Riyadh, Saudi Arabia: a retrospective analysis. BMC Emergency Medicine. 2024; 24: 64.

[9] Jafarizadeh A, Homaie M, Abdollahi M, Niyousha M. Time course study of optical coherence tomography angiography in patients with methanol induced optic neuropathy. BMC Ophthalmology. 2023; 23: 178.

[10] Sharma R, Marasini S, Sharma AK, Shrestha JK, Nepal BP. Methanol poisoning: ocular and neurological manifestations. Optometry and Vision Science. 2012; 89: 178–182.

[11] Waisberg E, Micieli JA. Neuro-ophthalmological optic nerve cupping: an overview. Eye and Brain. 2021; 13: 255–268.

[12] Tanrivermis Sayit A, Aslan K, Elmali M, Gungor I. Methanol-induced toxic optic neuropathy with diffusion weighted MRI findings. Cutaneous and Ocular Toxicology. 2016; 35: 337–340.

[13] Roberts DM, Yates C, Megarbane B, Winchester JF, Maclaren R,

Gosselin S, *et al*.; EXTRIP Work Group. Recommendations for the role of extracorporeal treatments in the management of acute methanol poisoning: a systematic review and consensus statement. Critical Care Medicine. 2015; 43: 461–472.

[14] Mower WR, Gupta M, Rodriguez R, Hendey GW. Validation of the sensitivity of the National Emergency X-Radiography Utilization Study (NEXUS) head computed tomographic (CT) decision instrument for selective imaging of blunt head injury patients: an observational study. PLOS Medicine. 2017; 14: e1002313.

[15] Kaewput W, Thongprayoon C, Petnak T, Chewcharat A, Boonpheng B, Bathini T, *et al*. Inpatient burden and mortality of methanol intoxication in the United States. The American Journal of the Medical Sciences. 2021; 361: 69–74.

[16] Aghababaeian H, Araghi Ahvazi L, Ostadtaghizadeh A. The methanol poisoning outbreaks in Iran 2018. Alcohol and Alcoholism. 2019; 54: 128–130.

[17] Güler S, Üçöz Kocaşaban D. An outbreak of home distillation methanol poisoning in turkey during the COVID-19 pandemic: a single center experience. Archives of Iranian Medicine. 2024; 27: 151–158.

[18] Gulen M, Satar S, Avci A, Acehan S, Orhan U, Nazik H. Methanol poisoning in Turkey: two outbreaks, a single center experience. Alcohol. 2020; 88: 83–90.

[19] Hovda KE, Hunderi OH, Rudberg N, Froyshov S, Jacobsen D. Anion and osmolal gaps in the diagnosis of methanol poisoning: clinical study in 28 patients. Intensive Care Medicine. 2004; 30: 1842–1846.

[20] Sasani MR, Molavi Vardanjani H, Mehdipour Namdar Z, Jeddi M, Seif S, Sedighi S, *et al*. Prognosis of methanol poisoning in a developing setting. Archives of Iranian Medicine. 2024; 27: 127–134.

[21] Alrobaian M, Alkhuraiji A, Almohanna R, Alshehri M, Alyahya B. Visual outcome of methanol toxic optic neuropathy after erythropoietin treatment in Riyadh, Saudi Arabia. Saudi Journal of Ophthalmology. 2023; 38: 41–46.

[22] Moretti R, Pizzi B. Ultrasonography of the optic nerve in neurocritically ill patients. Acta Anaesthesiologica Scandinavica. 2011; 55: 644–652.

[23] Geeraerts T, Launey Y, Martin L, Pottecher J, Vigué B, Duranteau J, *et al*. Ultrasonography of the optic nerve sheath may be useful for detecting raised intracranial pressure after severe brain injury. Intensive Care Medicine. 2007; 33: 1704–1711.

[24] Grzybowski A, Zülsdorff M, Wilhelm H, Tonagel F. Toxic optic neuropathies: an updated review. Acta Ophthalmologica. 2015; 93: 402–410.

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